CASE REPORT

Intractable vomiting due to a brainstem lesion in the absence of neurological signs or raised intracranial pressure

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Abstract

The case of a 30 year old man who was believed to have a gastrointestinal motility disorder causing his chronic vomiting is reported. He had been well until 21 months previously when he had developed recurrent vomiting which would occur up to 10 times in a 24 hour period. Vomiting was not precipitated by eating and was not associated with any other symptoms. He had lost 25 kg in weight. A psychiatric assessment did not reveal a psychogenic cause for his vomiting. A brainstem magnetic resonance imaging scan revealed an area of low signal in the low midbrain just above the pons to the left of the midline. After gadolinium contrast injection the area enhanced. There was little or no mass effect, that is minimal displacement of normal structures, and minimal oedema. The appearance was that of a low grade or early brainstem tumour. There were no features of haemorrhage or infarct. The patient was managed with oral dexamethasone, resulting in prompt resolution of his symptoms. A search for a central neurological cause is recommended in a patient with unexplained persistent vomiting, even in the absence of other features to suggest a neurological problem. Autonomic function testing may provide additional information.

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Vomiting is not usually an isolated symptom and may be a manifestation of underlying disease in the gastrointestinal tract, a systemic disease process, or rarely, may be due to psychogenic causes. The cause of the vomiting is usually apparent after clinical assessment and a few simple investigations. A neurological cause is rarely responsible; in cases where it is, even though vomiting may be the presenting symptom, this is almost invariably associated with either neurological symptoms or signs.

We report the case of a man who was believed to have a gastrointestinal motility disorder causing his chronic vomiting. Persistent

investigation ultimately revealed a small brain stem tumour. The patient had no other neurological symptoms or signs.

Case report

MR, aged 30, was referred for further investigation of unexplained vomiting. He had been well until 21 months previously when he had developed recurrent vomiting which would occur up to 10 times in a 24 hour period. The onset of symptoms coincided with his return from abroad. Vomiting was not precipitated by eating and was not associated with any other symptoms. He had lost 25 kg (four stone) in weight. A psychiatric assessment did not reveal a psychogenic cause for his vomiting.

Normal investigations had included a full blood count; biochemical profile; inflammatory markers; a chest x ray; barium meal and small bowel follow through; a small bowel enema; abdominal ultrasound scan; pelvic, abdominal, and cerebral computed tomography (CT) scans; gastroscopy; and colonoscopy.

He remained refractory to antiemetics and the promotility agents cisapride and erythromycin. His symptoms persisted despite trials of nasogastric feeding, total parenteral nutrition, and feeding via percutaneous gastrostomy tube. Eleven months after the onset of his illness a gastrojejunostomy and vagotomy was performed, but this also failed to alleviate his symptoms.

Eighteen months after the onset of his illness he was referred to our hospital. The sole symptoms were still intermittent unprovoked vomiting several times a day and a 25 kg (four stone) weight loss. The patient denied nausea. On direct questioning he admitted to a decreased bowel frequency of once per week, for over a year.

On examination he appeared well. General examination was completely normal apart from the abdominal scars from his surgery. Neurological examination was normal, including examination of the fundi, cranial nerves, and peripheral nervous system.

While on the ward the patient ate small quantities but would frequently vomit undigested food. Witnesses reported that this was not self induced. A dual radioisotope gastric emptying study demonstrated rapid transit of

Figure 1 Sagittal T1 weighted cerebral MRI scan showing an area of low signal in the low midbrain immediately above the pons.

Figure 2 Coronal T2 weighted cerebral MRI scan following gadolinium enhancement showing a lesion to the left of the midline which is associated with little mass effect and minimal oedema.

liquid to the terminal ileum, although he vomited the solid component of the meal within 5–10 minutes of ingestion.

A brainstem magnetic resonance imaging (MRI) scan was performed using a T1 weighted sequence in sagittal and coronal planes. This demonstrated an area of low signal in the low midbrain just above the pons to the left of the midline (fig 1). After gadolinium contrast injection the area enhanced (fig 2). There was little or no mass effect, that is minimal displacement of normal structures, and minimal oedema. The appearance was that of a low grade or early brainstem tumour. There were no features of haemorrhage or infarct.

Neurosurgical opinion was sought and it was felt that the tumour was surgically inaccessible, and that the risks associated with a stereotactic biopsy precluded any histological confirmation of the diagnosis. The patient was managed with oral dexamethasone, resulting in prompt resolution of his symptoms, such that his vomiting remains at bay after one year of follow up and he has gained weight.

Discussion

This case has shown that a very small lesion in the brainstem can specifically involve motility centres, causing profound symptoms such as

vomiting and decreased bowel frequency, in the absence of neurological signs. These tumours are likely to be slow growing and to elicit little oedema or shift of normal structures. Such a lesion should be sought when other pathology is not apparent and psychogenic factors do not appear to be important. This patient had had an earlier normal cerebral CT scan. Magnetic resonance imaging is superior in assessing the posterior fossa, and enhancing agents should be used.

To our knowledge only one similar case has been described in the literature. Wood *et al* described a 49 year old man with chronic nausea, anorexia, and vomiting due to a medullary glioma, who was initially thought to have an upper intestinal problem.¹ However that patient had other neurological symptoms: headache, hiccups, and yawning. Autonomic involvement was evidenced by orthostatic hypotension, impaired sweating, and altered cardiorespiratory reflexes. Efferent sympathetic and parasympathetic pathways were thought to be involved. Evidence for altered cerebral control of gut motility included slow propagation and incoordination of the migrating motor complexes on intestinal motility and prolonged gastric emptying.

Other causes of intracranial pathology can present with upper gastrointestinal symptoms alone. A 19 year old girl has been described who presented with a three week history of recurrent vomiting in the absence of a headache or neurological findings, due to a communicating hydrocephalus.² In another series four patients with posterior fossa mass lesions experienced protracted vomiting as their only symptom due to compression or displacement of the floor of the fourth ventricle by the lesion.³

The most interesting feature in our patient was the very selective involvement of centres responsible for vomiting and bowel transit. Studies performed in cats on the physiology of vomiting serve as a model for understanding vomiting in humans.^{4,5} They located a “vomiting centre” in the dorsal portion of the lateral reticular formation of the feline medulla. The reticular formation is a diffuse structure stretching from the superior cervical cord to the thalamic nuclei. The “vomiting centre” is anatomically and functionally separate from the chemosensitive trigger zone, which is in the area postrema in the floor of the fourth ventricle, but appears to act as the main relay centre for afferent and efferent impulses that are involved in the act of vomiting.

Vomiting can be triggered via stimulation of the chemosensitive trigger zone in the floor of the fourth ventricle by circulating emetogenic drugs such as opiates and dopamine agonists, but this is not the only source of input to the vomiting centre. Vagal afferents relay in the nucleus ambiguus, and other vagal and sympathetic afferents reach the nucleus tractus solitarius; all of these structures are closely related to the chemosensitive trigger zone and the vomiting centre. They are also linked to the vestibular system and corticobulbar fibres, which may also contribute to the development of vomiting.⁶

Normally the site of a brainstem lesion can be determined by localising neurological signs. Approximately one third of children with brainstem tumours have vomiting, often in the absence of raised intracranial pressure.^{7,8} In these patients vomiting usually occurs in association with cranial nerve or long tract signs, and may also be accompanied by autonomic disturbances or signs and symptoms of increased intracranial pressure. Squires recently reported on five children who had vomiting as the sole and initial symptom of an intracranial tumour.⁹ However this appears to be rare in adults.

Vomiting in our patient was unlikely to be due to raised intracranial pressure. There was no morning vomiting, no exacerbation with a change in posture, and no papilloedema. The likely explanation lies in the neuroanatomical relationship of the lesion to brainstem nuclei that provide vagal, motor, and autonomic efferents which specifically influence gastrointestinal motility.¹⁰⁻¹²

It is therefore necessary to look for a central neurological cause in a patient with unexplained persistent vomiting, even in the absence of other features to suggest a neurological problem. Autonomic function

testing may provide additional information. We believe that the investigation of choice is enhanced magnetic resonance imaging, and should be considered after exclusion of the more common causes.

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